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Abstracts

Symposium 2: Neuronal pathfinding and identity

Program/Abstract # 9**The form and function of an olfactory sensory map in the fly brain**

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Chemosensory cues play an important role in regulating social, courtship, and mating rituals in many different animals, communicating information about an individual's species, gender, reproductive status, genetic relatedness, and place in the social dominance hierarchy. This information is transmitted by pheromones, molecules produced by members of one species to influence the behavior of conspecifics. These compounds are detected by specialized gustatory or olfactory neurons. Pheromones were first described in insects but also influence behaviors in vertebrates. The fruit fly, *Drosophila melanogaster*, is an excellent model system to study chemosensory control of stereotyped behaviors because this animal has a simple olfactory system, exhibits stereotyped courtship behaviors, and can be genetically manipulated. Flies are generally attracted to food odors and repelled by carbon dioxide, which is a component of *Drosophila* stress odorant (dSO) released by stressed flies. Pheromonal cues are thought to elicit a number of stereotyped behaviors underlying courtship, aggression, aggregation, and other social behaviors. My group has been characterizing the odorant and gustatory receptors and circuits activated by pheromones, carbon dioxide, and food odors in order to achieve a comprehensive understanding of how this small insect interacts with its chemical environment. Recent work from my laboratory in elucidating the connectivity of the fly olfactory system, how it is activated by different odors, and how these odors mediate stereotyped behaviors will be presented.

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Program/Abstract # 10**Specification, migration, and differentiation of the left-sided parapineal organ**

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We seek to understand how left–right (L–R) differences arise during the development of the vertebrate brain. Significant advances have been made in zebrafish from studies of the epithalamus, which includes the left and right habenular nuclei, the pineal organ and the left-sided parapineal organ. A signal from the parapineal organ induces the left habenula to develop different anatomical and gene expression characteristics relative to the right habenula. Parapineal

cells originate in the middle of the brain but subsequently migrate leftward to lie adjacent to the left habenula. Time-lapse analysis reveals that parapineal cells move as a string of cells in close contact with one another. We have identified two genes, *tbx2b* and *fgf8*, as being required for parapineal development. The transcription factor *tbx2b* specifies the correct number of parapineal cells and allows them to coalesce and migrate leftward. Failure to migrate is not a secondary consequence of decreased cell number; after reducing the parapineal cell number by laser ablation in wild-type embryos, the remaining cells can still move to the left side of the brain. Subsequent to parapineal cell migration, *fgf8* is required for maintenance and/or differentiation of the parapineal cells. In contrast to other systems in which *fgf8* and *tbx2* regulate one another's activity, these two genes appear to act in separate pathways during parapineal development. Using genetic and embryological tools, three discrete steps (specification, migration, and differentiation) have been identified during the formation of an asymmetric brain structure.

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Program/Abstract # 11**Hypothyroidism-induced deafness: Defects in neuronal development and sensory cell function**M. Mustapha^{a,c}, Q. Fang^a, R.K. Duncan^a, Y. Raphael^b, D.F. Dolan^b, A. Giordimaina^b, T.-W. Gong, M. Lomax^b, K.R. Johnson^b, Sally A. Camper^b^a Department of Human Genetics, University of Michigan, Ann Arbor, MI, USA^b Department of Otorhinolaryngology, University of Michigan, Ann Arbor, MI, USA^c Jackson Laboratory, Bar Harbor, ME, USA

The absence of thyroid hormone during late gestation and early infancy can cause irreparable deafness and mental disability in both humans and rodents. To identify the underlying molecular mechanisms of hypothyroidism-induced hearing impairment, we exploited a mouse model of secondary hypothyroidism, (*Pou1f1*^{dw}), which has profound, congenital deafness due to failure of the pituitary gland to produce thyroid stimulating hormone. Thyroid hormone deficiency produces pleiotropic effects on cochlear development in these mice including some temporary features of developmental delay and numerous permanent defects that likely contribute to the profound deafness. These include impaired neuronal development and synapse formation, auditory sensory cell dysfunction and cell death, potassium channel gene expression abnormalities, and signs of cellular stress and premature aging. Cochlear innervation and synapse formation typically occurs in two phases during neonatal life, before and after the onset of hearing. Both short and long range guidance cues are